Mycobacterium gordonae genitourinary disease

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Abstract

***Mycobacterium gordonae* is frequently isolated from urine, but *M gordonae* genitourinary disease is rare; the majority of the isolates are commensals. We describe a 40 year old housewife who presented with loin pain, dysuria and frequency. Urine contained excessive pus cells, was sterile on culture and she did not respond to broad spectrum antibiotics. There was repeated isolation of *M gordonae* from the urine and she responded to a standard antituberculosis regimen. She was irregular and non-compliant with supervised therapy and relapsed three months after stopping medications. She again had symptoms and *M gordonae* was repeatedly isolated from the urine, *Mycobacterium tuberculosis* and other pathogens were not isolated. There was no evidence of humoral or cellular immunodeficiency or HIV infection.**

Introduction

Mycobacteria other than tubercule bacilli (MOTT) now account for an increasing variety of diseases in humans with the epidemic in human immunodeficiency virus (HIV) infections and the acquired immunodeficiency syndrome (AIDS).[1-3] This increase has been most marked in disseminated infections, soft tissue and pulmonary infections, while isolated genitourinary infection is much less common.[1-3] The increase in MOTT disease has been less with *Mycobacterium gordonae* infections, with little impact from the HIV and AIDS epidemics.[3] There are now well documented reports of *M gordonae* infections associated with skin, soft tissue and ocular diseases,[4 5] pulmonary disease,[6 7] prosthetic valve and shunt infections,[8 9 10] meningitis,[10] hepatic and peritoneal infections,[11] bone, joint and disseminated diseases.[8-12] Genitourinary *M gordonae* disease has been observed as part of a disseminated infection[12] but isolated genitourinary disease due to *M gordonae* is rare. Recent review of isolated MOTT genitourinary disease by Clark *et al*[13] which included a review of five publications, did not mention *M gordonae*; three cases had disease due to *M kansasii*; *M avium* intracellulare complex (MAIC) was isolated from three patients, with one case each of *M xenopei*, *M fortuitum* and *M chelonei* infection. The detailed review of non-tuberculous mycobacterial diseases by Wolinsky[1] included five cases of renal disease: two had *M kansasii* and three MAIC disease; there were three cases of epididymitis: one due to *M xenopei* and two others *M kansasii*. We report here a 40 year old housewife with genitourinary infection due to *M gordonae*.

Case report

A 40 year old housewife presented with persistent dysuria, frequency and left loin pain, which had not responded to broad spectrum antibiotic therapy. She was multiparous with eight children, had severe iron deficiency anaemia and had had abdominal tubal ligation following her last confinement 13 years earlier. Haemoglobin was 7·3 g/dl, serum iron 4·8 μmol/l and ferritin 3·2 μmol/l with high total iron binding capacity at 73·0 μmol/l. Anaemia had persisted as she did not take oral iron supplements regularly. Fasting blood sugar, urea and electrolytes were normal. Urine microscopy showed 80 WBC/HPF and 59 WBC/HPF, with colony count less than 10^4/ml and no growth on routine culture. Urine was negative on smear for acid-alcohol fast bacilli (AAFB), but gave preliminary growths of AAFB on BACTEC culture. Tuberculin test was strongly positive (28 mm, Mantoux) to five tuberculin units (PPD CT 68 strain). On review it was felt that the presentation of persistent dysuria, loin pain with sterile pyuria, in association with strongly positive tuberculin test and preliminary growth of mycobacteria on urine culture, was in keeping with renal tuberculosis and she was referred to the Tuberculosis Treatment Unit. She had cough productive of white sputum and evening fever. There was no history of previous treatment for tuberculosis, the chest radiograph was normal and sputum was negative on smear and culture for mycobacteria. Intravenous urography showed prompt excretion from normal size kidneys with normal pelvicalyceal pattern; there were no cortical scars or ureteric obstruction. ELISA for previous HIV infection was negative. Total immunoglobulins were IgA 298 mg/dl (70–312 mg/dl); IgE 28 kU/l (up to 200 kU/l); IgG 1540 mg/dl (639–1349 mg/dl) and IgM 185 mg/dl (56–352 mg/dl). Total lymphocyte count was 2·3 $\times 10^9$/l, with helper T4 lymphocytes 37·9% (31–55%); suppressor T8 lymphocytes 26% (17–38%) and normal T4/T8 ratio 1·4 (1·8 + 0·6).

She was commenced on combination antituberculosis regimen with ethambutol (E), isoniazid (H), rifampicin (R) and pyrazinamide (Z)–(EHRZ). The AAFB isolates were characterised as MOTT and were sent to Mayo Medical Laboratories for identification. She was difficult, irregular and non-compliant with daily supervised therapy at the Health Centre, so therapy was changed to EHRZ thrice weekly regimen at the Tuberculosis

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Treatment Unit after the third month. Dysuria persisted for sometime but she improved and urine became negative on smear and culture for mycobacteria. The MOTT isolates were subsequently identified as *Mycobacterium gordonae* (Mayo Medical Laboratories, Rochester, Minnesota). She became more regular and compliant but still complained about the medications. Treatment was stopped after nine months.

She was well, asymptomatic, urine had become negative on 14 smears and cultures for mycobacteria, and repeat intravenous urography showed normal kidneys, ureters and bladder. On follow-up at three months however, she again had dysuria with frequency and urine culture yielded *M gordonae* from two of three specimens. Repeat urine at six months also twice gave growths of *M gordonae*. She was called for review as "failed initial therapy" and after gynaecological review to exclude local pelvic pathology will be recommenced on therapy. Treatment failure here being related to initial difficulty and noncompliance with therapy. There was no evidence of humoral or cellular immunodeficiency.

Discussion

Genitourinary tuberculosis is often missed or diagnosed late with consequent destruction of renal tissue. Early disease is associated with abnormal findings on urinalysis, which commonly precede radiological evidence of renal disease.[14] There may be no gross urographic abnormality or minimal lesion with early disease, but advanced disease is associated with renal papillary and parenchymal damage with cavitation; followed by fibrosis, scarring and calcification on healing.[14-15] Where the initial urogram is normal, serial urograms should be done as later films may show abnormality.[14 16] The initial urogram in this case was normal and repeat examination after completing therapy was also normal, indicating mild early disease with no gross urographic abnormality.

The persistence of urinary symptoms associated with excessive pus cells in urine which is sterile on routine culture is highly suggestive of a tuberculous aetiology. The diagnosis is confirmed by the isolation of mycobacteria on culture. MOTT infections of the genitourinary tract not uncommonly follow instrumentation, trauma or surgery on the genitourinary tract.[13] This patient had abdominal sterilisation, bilateral tubal ligation, after delivery 13 years earlier.

Mycobacterium gordonae, previously known as the "tapwater bacillus", commonly isolated from water, and common in the environment, is one of the least pathogenic of the Runyon Group II scotochromogenic mycobacteria.[1 2] It has contaminated fluids in hospital practice accounting for nosocomial infections, and in other cases has been regarded as a non-pathogenic commensal.[17] Its isolation in a clinical setting, with symptoms suggestive of tuberculous disease, in the absence of *Mycobacterium tuberculosis* or other mycobacteria and likely pathogens, indicates a significant pathogenic role.[18] This patient presented with persistent dysuria, frequency and loin pain, did not respond to broad spectrum antibiotics, the urine had excessive pus cells with no growth on bacterial culture. *M tuberculosis* or other pathogens were not isolated, *M gordonae* was isolated from two out of three urine cultures. She responded well to therapy, though was non-compliant initially, and while on therapy with standard antituberculosis regimen, EHRZ, became asymptomatic and the urine became negative on smear and culture for mycobacteria on 14 occasions. Treatment was maintained for nine months only and considering that she was non-compliant for most of the initial three months of therapy, treatment may be judged as inadequate. Early relapse on follow up at three months, with symptoms and isolation of *M gordonae* from four of six urine specimens, indicating treatment failure due to inadequate therapy. The fact the urine was negative on culture $\times 14$ while on therapy, would suggest that the bacilli responded to and were sensitive to the regimen. There is evidence that the majority of *M gordonae* isolates are sensitive to rifampicin, with variable sensitivity to streptomycin and ethambutol, but uniform resistance to isoniazid. *M gordonae* disease usually shows good clinical response to regimens including rifampicin, inspite of the resistance to isoniazid.

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